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(FILE 'HOME' ENTERED AT 16:13:42 ON 07 JUL 2007)

FILE 'BIOSIS, EMBASE, MEDLINE' ENTERED AT 16:13:56 ON 07 JUL 2007

L1	1207	S ORGAN (S) ADHESION
L2	16	S L1 (S) ((PREVENT? OR PROPHYLAXSIS) (5A) ADHESION)
L3	9	S L2 NOT PD>20020220
L4	4	DUPLICATE REMOVE L3 (5 DUPLICATES REMOVED)
L5	7	DUPLICATE REMOVE L2 (9 DUPLICATES REMOVED)
L6	3	S L5 NOT L4

L4 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
TI Cerivastatin prevents angiotensin II-induced renal injury independent of blood pressure- and cholesterol-lowering effects.
AB Background: Statins are effective in prevention of end-organ damage; however, the benefits cannot be fully explained on the basis of cholesterol reduction. We used an angiotensin II (Ang II)-dependent model to test the hypothesis that cerivastatin prevents leukocyte adhesion and infiltration, induction of inducible nitric oxide synthase (iNOS), and ameliorates end-organ damage. Methods: We analyzed intracellular targets, such as mitogen-activated protein kinase and transcription factor (nuclear factor-kappaB and activator protein-1) activation. We used immunohistochemistry, immunocytochemistry, electrophoretic mobility shift assays, and enzyme-linked immunosorbent assay techniques. We treated rats transgenic for human renin and angiotensinogen (dTGR) chronically from week 4 to 7 with cerivastatin (0.5 mg/kg by gavage). Results: Untreated dTGR developed hypertension, cardiac hypertrophy, and renal damage, with a 100-fold increased albuminuria and focal cortical necrosis. dTGR mortality at the age of seven weeks was 45%. Immunohistochemistry showed increased iNOS expression in the endothelium and media of small vessels, infiltrating cells, afferent arterioles, and glomeruli of dTGR, which was greater in cortex than medulla. Phosphorylated extracellular signal regulated kinase (p-ERK) was increased in dTGR; nuclear factor-kappaB and activator protein-1 were both activated. Cerivastatin decreased systolic blood pressure compared with untreated dTGR (147 +- 14 vs. 201 +- 6 mm Hg, P < 0.001). Albuminuria was reduced by 60% (P = 0.001), and creatinine was lowered (0.45 +- 0.01 vs. 0.68 +- 0.05 mg/dL, P = 0.003); however, cholesterol was not reduced. Intercellular adhesion molecule-1 and vascular cell adhesion molecule-1 expression was diminished, while neutrophil and monocyte infiltration in the kidney was markedly reduced. ERK phosphorylation and transcription factor activation were reduced. In addition, in vitro incubation of vascular smooth muscle cells with cerivastatin (0.5 mumol/L) almost completely prevented the Ang II-induced ERK phosphorylation. Conclusion: Cerivastatin reduced inflammation, cell proliferation, and iNOS induction, which led to a reduction in cellular damage. Our findings suggest that 3-hydroxy-3-methylglutaryl coenzyme (HMG-CoA) reductase inhibition ameliorates Ang II-induced end-organ damage. We suggest that these effects were independent of cholesterol.
ACCESSION NUMBER: 2000:468358 BIOSIS
DOCUMENT NUMBER: PREV200000468358
TITLE: Cerivastatin prevents angiotensin II-induced renal injury independent of blood pressure- and cholesterol-lowering effects.
AUTHOR(S): Park, Joon-Keun; Mueller, Dominik N.; Mervaala, Eero M. A.; Dechend, Ralf; Fiebeler, Anette; Schmidt, Folke; Bieringer, Markus; Schaefer, Olaf; Lindschau, Carsten; Schneider, Wolfgang; Ganten, Detlev; Luft, Friedrich C. [Reprint author]; Haller, Hermann
CORPORATE SOURCE: Franz Volhard Clinic, Wiltberg Strasse 50, 13125, Berlin, Germany
SOURCE: Kidney International, (October, 2000) Vol. 58, No. 4, pp. 1420-1430. print.
CODEN: KDYIA5. ISSN: 0085-2538.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Nov 2000
Last Updated on STN: 10 Jan 2002

L4 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Method for reducing or preventing post-surgical adhesion formation using ketotifen and analogs thereof.
AB Compositions and methods for minimizing or preventing

post-surgical adhesion formation between tissue, e.g., organ, surfaces in body cavities, whereby an effective therapeutic amount of anti-asthmatic ketotifen (4-1(1-methyl-4-piperidyliden-4H-benzo[4,5]cyclohepta[1,2-b] thiophene-10 (9H)-one, hydrogen fumarate salt) thereof is administered to the target injury site for a period of time sufficient to permit tissue repair. Ketotifen or analogs thereof is preferably administered in conjunction with a delivery vehicle (e.g., microcapsules, microspheres, biodegradable polymer films, lipid-based delivery systems such as liposomes and lipid foams, crystalloid and viscous instillates and absorbable mechanical barriers) useful for maintaining local concentrations of the inhibitor at the injury site at an effective level for a sustained period of time.

ACCESSION NUMBER: 1999:305450 BIOSIS
DOCUMENT NUMBER: PREV199900305450
TITLE: Method for reducing or preventing post-surgical adhesion formation using ketotifen and analogs thereof.
AUTHOR(S): Dizerega, Gere Stodder [Inventor]; Rodgers, Kathleen Elizabeth [Inventor, Reprint author]
CORPORATE SOURCE: Miller's Children's Hospital of Long Beach, Long Beach, CA, USA
ASSIGNEE: University of Southern California University Park Campus
PATENT INFORMATION: US 5891460 19990615
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (15-JUN-99) Vol. 1221, No. 1. print.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Aug 1999
Last Updated on STN: 12 Aug 1999

L4 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 2
TI Nitric oxide modulation of transcellular biosynthesis of cys-leukotrienes in rabbit leukocyte-perfused heart.
AB 1. We have studied the role of nitric oxide (NO) in the regulation of the transcellular biosynthesis of sulphidopeptide leukotrienes (cys-LT) generated upon neutrophil-vascular wall interactions and their functional consequences, in the spontaneously beating, cell-perfused, heart of the rabbit. 2. Hearts were perfused under recirculating conditions (50 ml) with 5 times 10-6 purified human neutrophils (PMNL), and challenged with 0.5 mu-M A-23187 for 30 min. Coronary perfusion pressure (CPP) and left-ventricular end-diastolic pressure (LVEDP) were monitored. Cys-LT formation was measured by reversed phase high performance liquid chromatography (h.p.l.c.) and u.v. spectral analysis. Myeloperoxidase (MPO) enzyme activity, assayed in aliquots of the recirculating buffer, was used as a marker of PMNL adhesion to the coronary endothelium. 3. Basal CPP and LVEDP values averaged 45 +- 1.4 mmHg and 5 +- 0.1 mmHg, respectively; A-23187 triggered an increase in CPP (134 +- 9 mmHg, at 30 min) which was significantly attenuated by pretreatment with L-arginine, 100 mu-M (90 +- 3 mmHg, at 30 min). Pretreatment with N-G-monomethyl-L-arginine, 10 mu-M (L-NMMA), induced a marked increase in CPP (290 +- 40 mmHg, at 20 min) and in LVEDP (47 +- 16 mmHg), so pronounced that it caused cardiac arrest in systole in 5 out of 6 hearts and these were prevented by L-arginine, 100 mu-M (CPP 115 +- 10 mmHg, LVEDP 6 +- 1.1 mmHg, at 30 min). 4. The increase in CPP was accompanied by the release of cys-LT in the circulating buffer, which was reduced significantly by L-arginine. Pretreatment with L-NMMA, caused a marked rise in cys-LT concentrations which was prevented by L-arginine. 5. Neither L-arginine nor L-NMMA affected directly the A-23187-induced arachidonic acid (AA) metabolism in isolated PMNL alone. 6. Pretreatment with L-NMMA caused a prompt drop in myeloperoxidase (MPO) activity, suggesting rapid adhesion of PMNL to the coronary wall; this effect was significantly blunted by L-arginine. 7. This study suggests that NO

provides cardioprotection in an organ model of transcellular metabolism of cys-LT by preventing PMNL adhesion to the coronary intima.

ACCESSION NUMBER: 1997:200559 BIOSIS
DOCUMENT NUMBER: PREV199799499762
TITLE: Nitric oxide modulation of transcellular biosynthesis of cys-leukotrienes in rabbit leukocyte-perfused heart.
AUTHOR(S): Buccellati, Carola; Rossoni, Giuseppe; Bonazzi, Albino; Berti, Ferruccio; Maclouf, Jacques; Folco, Giancarlo [Reprint author]; Sala, Angelo
CORPORATE SOURCE: Cent. Cardiopulmonary Pharmacol., Inst. Pharmacol. Sci., Via Balzaretti 9, 20133 Milan, Italy
SOURCE: British Journal of Pharmacology, (1997) Vol. 120, No. 6, pp. 1128-1134.
CODEN: BJPCBM. ISSN: 0007-1188.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 May 1997
Last Updated on STN: 12 May 1997

L4 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 3

TI Do plasma levels of circulating soluble adhesion molecules differ between surviving and nonsurviving critically 11 patients?.

AB Adhesion molecules appear to play a central role in tissue damage secondary to inflammatory response. Besides various neutrophil- and endothelial-bound adhesion molecules, soluble forms of endothelial-derived adhesion molecules have been detected in the circulating blood in recent years. They seem to be good markers of endothelial damage, but their importance in the critically ill has not been definitely elucidated yet. Plasma levels of circulating (soluble) adhesion molecules (endothelial leucocyte adhesion molecules (sELAM-1), vascular cell adhesion molecule 1 (sVCAM-1), intercellular adhesion molecule 1 (sICAM-1)) were serially measured from arterial blood samples using enzyme-linked immunosorbent assays (ELISA) in 50 consecutive patients suffering from severe trauma (injury severity score (ISS) gt 25 points) or postoperative complications. Measurements were carried out on the day of admission on the intensive care unit (ICU) ("baseline" value) and during the next 5 days. Survival was defined as survival throughout the study period. The survivor group (n=30) consisted of more patients who had sustained trauma (53%), whereas in the nonsurvivors (n=20) more patients with postoperative complications were found (65%). On admission to ICU, septic shock was more often seen in the nonsurvivors (30%) than in the survivors (13%) and the nonsurvivors showed a slightly higher APACHE II score at baseline. At baseline, plasma levels of all three adhesion molecules were elevated beyond normal range in both groups. The sICAM-1 and sELAM-1 plasma concentrations were significantly higher in the nonsurvivors than in the survivors already at baseline. The sELAM-1 and sICAM-1 values significantly decreased in the survivors without reaching normal values. At the end of the investigation period, sVCAM-1 plasma level was within normal range in the survivors. In the nonsurvivors, all three adhesion molecules increased significantly throughout the study period (sELAM-1, from 115 +- 31 to 158 +- 23 ng/mL; sICAM-1, from 830 +- 210 to 1,536 +-199 ng/mL; sVCAM-1, from 861 +- 168 to 1,249 +- 151 ng/mL). None of the other hemodynamic or laboratory variables could be correlated with the time course of adhesion molecules, except for PaO-2/PaO-2 ratio, which was negatively correlated with plasma levels of soluble adhesion molecules in the nonsurvivors (analysis of covariance). It is concluded that plasma levels of soluble adhesion molecules were markedly higher in nonsurviving than in surviving critically ill patients. They may possibly serve as markers of the extent of inflammatory response, of the endothelial damage in patients at risk of multiple-organ failure or both. Their role in critical illness, however, is not definitely clarified. Thus, it has to be elucidated whether preventing an increase in soluble adhesion molecules is

of benefit for the patient's organ function or even outcome.

ACCESSION NUMBER:	1995:210346 BIOSIS
DOCUMENT NUMBER:	PREV199598224646
TITLE:	Do plasma levels of circulating soluble adhesion molecules differ between surviving and nonsurviving critically 11 patients?.
AUTHOR(S):	Boldt, Joachim; Wollbrueck, Matthias; Kuhn, Detleft; Linke, L. Christoph; Hempelmann, Gunter
CORPORATE SOURCE:	Dep. Anesthesiology Intensive Care Med., Justus-Liebig-Univ. Giessen, Giessen, Germany
SOURCE:	Chest, (1995) Vol. 107, No. 3, pp. 787-792. CODEN: CHETBF. ISSN: 0012-3692.
DOCUMENT TYPE:	Article
LANGUAGE:	English
ENTRY DATE:	Entered STN: 23 May 1995 Last Updated on STN: 23 May 1995

L6 ANSWER 1 OF 3 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Hydrogel thin film containing extracellular matrix components.
AB The thin film of the invention comprises a hydrate of a vitrified gel containing one or more extracellular matrix components, which can be integrated with a retainer as required. A hydrogel thin film containing one or more extracellular matrix components such as thin-film collagen hydrogel thin film, which is useful for a cell culture substratum and for preventing organ adhesion, can be easily prepared, and is excellent in expediency.
ACCESSION NUMBER: 2007:243613 BIOSIS
DOCUMENT NUMBER: PREV200700242082
TITLE: Hydrogel thin film containing extracellular matrix components.
AUTHOR(S): Anonymous; Takezawa, Toshiaki [Inventor]; Yoshizato, Katsutoshi [Inventor]
CORPORATE SOURCE: Hyogo, Japan
ASSIGNEE: Japan Science and Technology Corporation
PATENT INFORMATION: US 07195912 20070327
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (MAR 27 2007)
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Apr 2007
Last Updated on STN: 11 Apr 2007

L6 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI No role of alpha-Gal in human monocyte-endothelial cell interactions in vitro.
AB Vascularized organ xenografts undergoing acute vascular rejection (AVR) are infiltrated by innate immune cells such as monocytes/macrophages. Herein, human monocyte static and dynamic adhesion to, and migration across, human and porcine aortic endothelial cells (HAEC and PAEC) were investigated. To elucidate the role of Gal alpha 1,3Gal (alpha-Gal) epitopes in these processes in the absence of anti-Gal antibodies (Ab), this determinant was aberrantly expressed in HAEC. HAEC were transduced with a lentiviral vector encoding the porcine alpha 1,3 galactosyltransferase to express alpha-Gal at high frequencies (75-95%). alpha-Gal expression on HAEC did not increase their ability to support monocyte transendothelial migration or adhesion under either static or flow conditions. Porcine and human endothelium supported static adhesion and migration of monocytes equally well. However, human monocytes adhered less to PAEC than to HAEC ($P = 0.03$) under flow following human, but not porcine, turnout necrosis factor-alpha stimulation. In the absence of anti-Gal Ab, the alpha-Gal epitope does not contribute to increased monocyte adhesion to, or migration across, endothelium. Thus, inhibiting adhesion receptor-ligand interactions essential for the adhesion of human monocytes to porcine endothelium may be more important than carbohydrate remodelling of donor pigs to prevent adhesion/infiltration of monocytes into organ xenografts during AVR.
ACCESSION NUMBER: 2006:110366 BIOSIS
DOCUMENT NUMBER: PREV200600109817
TITLE: No role of alpha-Gal in human monocyte-endothelial cell interactions in vitro.
AUTHOR(S): Ehrnfelt, C.; He, Z.; Holgersson, J. [Reprint Author]
CORPORATE SOURCE: Karolinska Univ Hosp Huddinge, Div Clin Immunol F79, Karolinska Inst, S-14186 Huddinge, Sweden
jan.holgersson@labmed.ki.se
SOURCE: Scandinavian Journal of Immunology, (NOV 2005) Vol. 62, No. 5, pp. 445-452.
CODEN: SJIMAX. ISSN: 0300-9475.
DOCUMENT TYPE: Article

LANGUAGE: English
ENTRY DATE: Entered STN: 8 Feb 2006
Last Updated on STN: 8 Feb 2006

L6 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Ketotifen abrogates local and systemic consequences of rat intestinal ischemia-reperfusion injury.
AB Background: Mast cell-derived vasoactive and pro-inflammatory mediators, particularly histamine, might contribute to local tissue damage and multiorgan dysfunction induced by intestinal ischemia/reperfusion (I/R). The purpose of the present study was to evaluate the effects of the mast cell stabilizer, ketotifen, on leukocyte adhesion within, and tissue leakage from the mucosal villous microcirculation after intestinal I/R.Methods: Superior mesenteric arteries of untreated and ketotifen-pretreated (1 mg/kg orally twice daily for 3 days, and 90 min prior to ischemia) Piebald-Viral-Glaxo (PVG) rats were clamped for 30 min (n = 12 per group; sham operated controls n = 12). Mucosal surfaces of exteriorized ileal segments were visualized, and leukocyte adherence in, and macromolecular leakage (MML) from individual villi were followed for 2 h after clamp removal using in vivo microscopy. Blood pressure and heart rate were monitored, and lung tissue damage was assessed by histology.Results: Ten untreated animals subjected to intestinal I/R failed to survive the reperfusion period, leukocyte adhesion ($P < 0.001$) and MML ($P < 0.001$) were increased at all time-points and blood flow stasis eventually ensued. In contrast, all ketotifen-pretreated I/R animals survived the duration of the study. Ketotifen abrogated I/R-induced leukocyte adherence within the villus mucosal capillaries and supplying arterioles and largely prevented pulmonary injury, yet surprisingly had no effect on intestinal vascular leakage.Conclusions: This is the first study to demonstrate that ketotifen is a powerful inhibitor of I/R-induced leukocyte adhesion and can prevent localized and reduce remote organ damage after intestinal I/R injury. However, its effects are manifested in the absence of any influence on intestinal I/R-induced vascular leakage.(C) 2005 Blackwell Publishing Asia Pty Ltd.
ACCESSION NUMBER: 2005:348216 BIOSIS
DOCUMENT NUMBER: PREV200510139142
TITLE: Ketotifen abrogates local and systemic consequences of rat intestinal ischemia-reperfusion injury.
AUTHOR(S): Kalia, Neena; Brown, Nicola J. [Reprint Author]; Wood, Richard F. M.; Pockley, A. Graham
CORPORATE SOURCE: Royal Hallamshire Hosp, Div Clin Sci S, Acad Unit Surg Oncol, Floor K, Sheffield S10 2JF, S Yorkshire, UK
n.j.brown@sheffield.ac.uk
SOURCE: Journal of Gastroenterology and Hepatology, (JUL 2005) Vol. 20, No. 7, pp. 1032-1038.
CODEN: JGHEEO. ISSN: 0815-9319.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Sep 2005
Last Updated on STN: 8 Sep 2005